In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 15-860V
(to be published)

* *	Chief Special Master Corcoran
REGINA TODD, *	
*	Filed: January 8, 2020
Petitioner, *	
*	
v. *	
*	Influenza Vaccine; Small Fiber
SECRETARY OF HEALTH AND *	Neuropathy; Skin Biopsy; Expert
HUMAN SERVICES, *	Testimony
*	
Respondent. *	
*	
* *	

Amber Wilson, Maglio, Christopher & Tolle, P.C., Washington, D.C., for Petitioner.

Debra A. Filteau Begley, U.S. Dep't of Justice, Washington, D.C., for Respondent.

DECISION[1]

On August 12, 2014, Regina Todd filed a petition seeking compensation under the National Vaccine Injury Compensation Program ("Vaccine Program")[2] based on the assertion that she developed a neuropathy (alleged at hearing to be properly diagnosed as a non-length dependent small fiber neuropathy ("NLDSFN")) after receiving the influenza ("flu") vaccine on September 17, 2012. Petition ("Pet.") (ECF No. 1) at 1. An entitlement hearing was held on March 4–5, 2019, in Washington, D.C., and the parties subsequently filed post-hearing briefs and supplemental reports, completing that process in July 2019.

[1] This Decision shall be posted on the Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012)). **This means that the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

The matter is finally ripe for resolution, and after review of the record and all submissions, I deny an entitlement award in this case. As discussed in greater detail below, Petitioner has not successfully established that she more likely than not experienced NLDSFN, or more broadly that the various symptoms she alleges were the product of a vaccine-initiated injury.

I. Factual Background

A. *Vaccination and Initial Symptoms*

Ms. Todd (a full-time nurse) received the flu vaccine on September 17, 2012. *See* Ex. 1; Tr. at 8. At the time, she was in good health, although she had previously been treated for type 2 diabetes. Ex. 8 at 19–20. Approximately three weeks later, on October 8, 2012, Petitioner went to her primary care provider, Thomas Crosby, M.D., complaining of a three-week history of intermittent "sciatica" symptoms and cramping in one leg, as well as a burning sensation in her left leg since the prior Saturday. *Id.* at 19. The records from this visit do not associate these symptoms with the vaccination Petitioner had received the prior month. Dr. Crosby diagnosed her with sciatic neuritis and muscle spasms. *Id.*

At the end of that month, on October 31, 2012, Ms. Todd saw an orthopedist, Mark Triana, D.O., for persisting symptoms associated with those she had initially reported to Dr. Crosby. Ex. 2 at 2. At the time of this visit, she specifically told Dr. Triana that her symptoms first began four weeks before (meaning the first week of October) as a "burning pain in her left buttock," that resolved but then returned the following week, with the sensation radiating down her left leg and into her foot, along with a constellation of other new symptoms (tingling, weakness, skin sensitivity akin to a sunburn, and burning feelings) in her right buttock, feet, and arms. *Id.*

A physical exam documented normal strength and reflexes. Ex. 2. at 2–3. Testing ordered by Dr. Triana revealed normal erythrocyte sedimentation rate ("ESR") and C-reactive protein ("CRP"), both well-recognized biomarkers establishing the presence of systemic inflammation. Ex. 6 at 20, 22. Rheumatoid Factor ("RF") and anti-nuclear antibody ("ANA")[3] testing also produced negative results. *Id.* at 24–25. Dr. Triana assessed Ms. Todd with a disturbance of skin sensation, "unspecified hereditary and idiopathic peripheral neuropathy," joint pain, and lumbago, and he prescribed medication. Ex. 2 at 3. A lumbar x-ray and MRI performed the next day (November 1, 2012) were both normal. *Id.* at 2–3, 6.

B. *2012 Hospitalization*

Within a week of her visit to Dr. Triana, Ms. Todd was admitted to Waccamaw Community Hospital in Murrells Inlet, South Carolina, on November 6, 2012, due to concerns about the progression of the symptoms she reported having experienced over the prior five weeks. Ex. 7 at

[3] An antinuclear antibody ("ANA") test is typically used to assess the presence of Systemic lupus erythematosus ("SLE"), and other autoimmune diseases. Because healthy individuals often test positive for ANA, however, follow-up testing is necessary to corroborate the diagnosis of an autoimmune disease, thus a negative result typically excludes the diagnosis of some autoimmune diseases or an ongoing autoimmune process. *See* K. Pagana et al., *Mosby's: Manual of Diagnostic and Laboratory Tests* 80 (6th ed. 2018) (hereinafter "*Mosby's*").

4–8, 10–11. She remained hospitalized (albeit at more than one facility) over the next five days. Ex. 10 at 85–86.

As Petitioner recounted during an initial assessment at Waccamaw by a neurologist, Paul Amodeo, M.D., the symptoms course she had been experiencing began in the weeks after receiving the flu vaccine, and had progressed to burning pain in both her legs and pelvis that spread to her arms, chest, back, and shoulders. Ex. 7at 5. The week immediately before seeking hospitalization, she began to feel unsteady when walking, worsening to the point where she could not walk at all. *Id.* Before her admission, Petitioner had sought treatment in the ER on November 5, 2012, and was administered 60 milligrams of prednisone, but felt that she had not improved with that medication. *Id.*

On exam, Ms. Todd's strength, reflexes, and sensation were normal. Ex. 7 at 6–7. In addition, although Petitioner reported hyperesthesia[4] to light touch across her shoulders and along the dorsal aspect of her arms, her sensation was intact to pinprick and vibratory testing. *Id.* at 7. And even though Petitioner reported difficulty standing, in Dr. Amodeo's assessment her complaints were "out of proportion with her strength to confrontation testing." *Id.* at 8. He thus concluded that the "etiology for the patient's presentation is unclear," but ordered further testing to evaluate a number of possible diagnoses, including a cervical cord pathology, Guillain-Barré syndrome ("GBS"), a small fiber neuropathy, or dorsal root ganglionopathy. *Id.* The subsequent testing (which included an MRI of Petitioner's cervical spine and brain, a cerebrospinal fluid ("CSF") study, and several autoimmune labs) produced uniformly normal results. *Id.* at 14, 75–81, 83; Ex. 8 at 41.

While at Waccamaw, Petitioner was initially treated with a course of prednisone, but the medication was discontinued as unnecessary given the absence of evidence of ongoing systemic inflammation (as reflected in normal CRP and ESR test results). Ex. 7 at 3. On November 8, 2012 (two days after admission), Ms. Todd was transferred to Medical University of South Carolina ("MUSC") with a discharge diagnosis of generalized weakness and a disturbance of skin sensation. *Id.* at 2. The discharging physician reported his view that her symptoms could be related to her flu vaccine, but he ultimately deferred to Petitioner's treating neurologists. *Id.*

Thereafter, Ms. Todd was evaluated at MUSC by Paul Pritchard, M.D., a neurologist. Ex. 10 at 103. Upon arrival, it was noted that she was tired and unwilling to fully engage in an examination (possibly because of a sedative she had received just prior). *Id.* at 109. Under such circumstances, Dr. Pritchard was only able to observe that her findings were "limited to subtle changes consistent with a L5-S1 radiculopathy." *Id.* at 111. However, an EMG completed on November 9, 2011, showed no signs of radiculopathy or neuropathy. *Id.* at 108. Based on Petitioner's overall presentation and testing, Dr. Pritchard concluded that there was no evidence of a neuropathic process, and started her on an antidepressant. *Id.* at 180–81.

[4] Hyperesthesia is "a dysesthesia consisting of increased sensitivity, particularly a painful sensation from a normally painless touch stimulus." *Dorland's Illustrated Medical Dictionary* 888 (32 ed. 2012) (hereinafter "*Dorland's*").

Petitioner subsequently improved and did not require any physical therapy. Ex. 10 at 85. At discharge on November 11, 2012, Dr. Pritchard noted that Ms. Todd's "neurological examination was not consistent . . . [t]here were no obvious sensory deficits . . . [t]here were no obvious motor deficits," and there was no clear basis for her symptoms. *Id.* at 85. The discharge diagnoses included "possible viral syndrome" and "lumbar radiculopathy," but made no mention of other neuropathic injury, whether caused by the flu vaccine or otherwise. *Id.* at 86. Nevertheless, Petitioner's primary care physician, Dr. Crosby, noted at a follow-up visit on November 19, 2012, that Ms. Todd might have "a mild case of [GBS]" (although the medical record containing this notation does not explain the basis for his opinion), and he recommended against her receiving the flu vaccine in the future. Ex. 8 at 18.

C. *2013 Treatment*

There is a subsequent five-month gap in the medical records, during which time Ms. Todd appears not to have experienced any recurrent symptoms associated with her fall 2012 hospitalization. On April 16, 2013, however, Petitioner called Dr. Pritchard's office reporting that her symptoms had reappeared about one week earlier, particularly characterized by a burning sensation when touched in certain parts of her body. Ex. 10 at 3. The following month, Petitioner saw Dr. Crosby on May 20, 2013, and reported a burning pain in her back, although she also added that her buttock and leg pain had resolved. Ex. 8 at 13.

Later that summer, in July 2013, Ms. Todd was evaluated by infectious disease specialist Joseph Cantley, M.D. Ex. 10 at 6. She reported waxing and waning pain symptoms since she received the flu vaccine in September 2012, plus a transient rash cluster on her right or left side. *Id.* at 6–7. Dr. Cantley's examination, however, revealed normal reflexes, normal sensation (based on a vibration test on her upper and lower extremities), and a normal gait. *Id.* at 8. Dr. Cantley concluded that Petitioner's symptoms were inconsistent with a peripheral neuropathy, and "unlikely" to be attributable to a vaccine reaction, preferring anxiety as an explanation. *Id.*

In the second half of that month, Petitioner again saw Dr. Pritchard, and informed him of the symptoms flares she claimed to have been experiencing since the spring, plus rashes and fatigue. Ex. 10 at 10. Although examination revealed a slightly awkward gait, Petitioner also appeared to possess normal strength, normal reflexes, and normal sensory test results. *Id.* Dr. Pritchard thus opined that Ms. Todd had "diffuse paresthesias and dysesthesias for which I cannot determine a cause or anatomical locus." *Id.* at 11. He suggested she restart antidepressants, or consider a consultation with a neuromuscular specialist. *Id.* Petitioner also saw a dermatologist that August, who (after examination, culture testing, and a biopsy) opined that her intermittent rash was more likely attributable to dry skin than to a cutaneous autoimmune condition. Ex. 5 at 2–3, 5–6.

Acting on Dr. Pritchard's recommendation, Ms. Todd saw a neuromuscular specialist, David Stickler, M.D., in September 2013. Ex. 10 at 12. Dr. Stickler's examination revealed both normal reflexes and intact "sensory modalities." *Id.* at 15. Given Petitioner's reported history, Dr. Stickler nevertheless allowed for the possibility that she might have a migratory sensory neuritis

or small fiber neuropathy. However, additional testing to confirm this speculation revealed a negative ANA of 1:80, a normal CRP, and negative antibodies for a variety of infectious processes. *Id.* 15, 248. Based upon such negative testing results, Dr. Stickler rejected his earlier speculation about the etiology of Petitioner' condition, adding that he could provide no further treatment for it. *Id.* at 16.

D. *2014 Treatment*

Ms. Todd maintains that she continued into 2014 to suffer from intermittent flares of the condition that purportedly began with her September 2012 vaccination, although the medical records suggest only sporadic treatment occurrences. For example, in March 2014, Petitioner informed Dr. Jeffrey Bush, M.D. at MUSC that her symptoms now encompassed her face and included difficulty swallowing. Ex. 10 at 19. Testing, however, again revealed a lack of inflammation biomarkers and no disease-associated antibodies. Ex. 6 at 42–43. Ms. Todd also had an emergency room visit on March 26, 2014, but her exam was largely normal, and a urinary tract infection was proposed as the most likely source of her symptoms flare. *Id.* at 21–23, 247. Indeed, while Petitioner was in the ER, Dr. Pritchard (her MUSC neurologist) was contacted by current treaters, and he explained to them that he had previously been unable to propose or identify a neurologic cause for her symptoms. *Id.* at 27. Dr. Pritchard directly told Ms. Todd the same thing at a return visit to him on April 1, 2014, referring her instead to a rheumatologist. Ex. 8 at 2.

Later that same April, Petitioner sought a second opinion from Dr. Fletcher Hartsell, M.D., a neuroimmunologist at Duke University's Health System. Ex. 9 at 17. His examination, however, revealed normal results, including sensory response. *Id.* at 19. Dr. Hartsell assessed Ms. Todd with diffuse pain and disturbance of her skin sensation, adding that her symptoms might be consistent with several different underlying conditions: central pain syndrome, SFN, or fibromyalgia. To evaluate if any could be the proper explanation for Petitioner's constellation of symptoms, Dr. Hartsell ordered a skin biopsy and additional laboratory testing. *Id.* at 20. But such testing again produced normal results—other than a positive ANA that persisted, as reflected in subsequent testing from May. *Id.* at 8, 32, 99.

The most recent medical records filed in this matter are from August 2014, around the time of the case's initiation. At the end of that month, Ms. Todd was evaluated by Sara Wasserman, M.D., a rheumatologist. Ex. 12 at 4. Dr. Wasserman's examination noted many tender points on Petitioner's back, upper and lower shoulders, arms, and thighs, but no joint tenderness, and Petitioner's strength and reflexes were normal as before. *Id.* at 7. Although Dr. Wasserman took note of Petitioner's positive ANA, she deemed it a nonspecific finding, present in a significant minority of the population. *Id.* at 8. Dr. Wasserman ultimately could not provide an etiology for Petitioner's condition, and did not deem it reflective of a connective tissue disease. *Id.*

That same month, Ms. Todd saw Thomas Buchheit, M.D., a pain management specialist,

who (taking into account the fact that testing did not suggest an autoimmune process at work) assessed her with probable fibromyalgia. Ex. 8 at 32. Petitioner was thereafter treated with a combination of medications based on a working diagnosis of fibromyalgia. *See* Ex. 13 at 2, 24. This, plus physical therapy, has led her to report improvement in her pain symptoms. *Id.* at 26. She had not, however, followed up with any neurologists since 2015. Tr. at 29.

II. Witness Testimony

A. *Ms. Todd*

Petitioner's testimony was largely consistent with the filed medical record. *See generally* Tr. at 8–31. She emphasized her good health prior to receipt of the flu vaccine—in contrast to the sensations of burning and skin sensitivity she purports to have experienced the month after. *Id.* at 14–15. She recounted the circumstances of her visit to the ER at Waccamaw Hospital and subsequent admission, detailing the weakness, pain, and other seeming neuropathic symptoms she was experiencing, along with how frightening she found the experience. *Id.* at 15–19. Thereafter, despite some improvement, Ms. Todd felt that she never returned to baseline, noting that cessation of her medication could result in a dramatic relapse. *Id.* at 19–20. She agreed, however, that treaters never were able to pinpoint an explanation for her condition, and that she never received a diagnosis of small fiber neuropathy, although she maintained that treaters had speculated that some of her symptoms resembled GBS. *Id.* at 20–21, 29–31.

Some of Ms. Todd's testimony was intended to expand on the impact her alleged vaccine injury has had on her life. Thus, she has been unable to complete certain training she had been participating in to expand her professional capabilities as a nurse (which in turn would have permitted an increase in income). Tr. at 10–11. She has had to seek accommodations at work that allow her to rest frequently and/or avoid standing for extended periods of time. *Id.* at 22–23. Her ability to engage in leisure activities has also been curtailed due to the burning sensations she experiences if seated for too long. *Id.* at 23–24.

B. *Petitioner's Expert – Dr. Enrique Aradillas-Lopez*

Dr. Aradillas prepared a single expert report in this case and testified at hearing. *See* Report, dated February 27, 2017, filed as Ex. 14 (ECF No. 41-2); Tr. at 33–190. He opined generally that Ms. Todd's chronic pain, which he deemed neuropathic in nature, was attributable to an adverse immunologic reaction produced by her September 2012 flu vaccine. Tr. at 76; Aradillas Rep. at 5.

As elaborated upon in his filed CV, Dr. Arradillas is a board-certified neurologist specializing in pain management. Tr. at 34, 43; CV, filed as Ex. 15 on Feb. 27, 2017 (ECF No. 41-3) ("Arradillas CV"). He does not hold board certification in immunology. Tr. at 157. Previously, he served as an assistant professor in the Drexel University College of Medicine Department of Neurology Pain Division. Arradillas CV at 1. Dr. Arradillas received his medical degree from La Salle University School of Medicine in Mexico City, Mexico. *Id.* Thereafter, he completed his

residency in internal medicine at Interfaith Medical Center in Brooklyn, New York as well as a residency in neurology at Drexel University College of Medicine in Philadelphia, Pennsylvania. *Id.* He then completed an interventional pain management fellowship at the Milton S. Hershey Medical Center in Hershey, Pennsylvania. *Id.*

The overwhelming majority of Dr. Aradillas's patients seek treatment from him for complex regional pain syndrome ("CRPS") or small fiber neuropathy, and only a small percentage of his clinical time is spent treating patients on a regular neurology service. Tr. at 87, 160. In addition to his clinical work, Dr. Arradillas has conducted research related to CRPS and has published several articles on the subject. *Id.* at 2–5. His publications, however, do not reflect research on the subjects of immunology or immunological responses to vaccination or disease. Tr. at 153–54.

At the outset of his testimony, Dr. Aradillas began with a general explanation of pain comparable to what Ms. Todd had experienced. He termed pain an "unpleasant sensory and emotional experience" that needs to be taken seriously when reported to treaters by a patient. Tr. at 42–43. He initially defined two kinds of pain: "neuropathic" pain attributable to injury or nervous system disease, and "somatic" pain (although he did not clearly explain how that truly differed from the first, and later expanded the field to include other categories). *Id.* at 44–45. Relying on the patient's description, a treater needs to try to ascertain the pain's source for treatment purposes. *Id.* at 43–44. However, Dr. Aradillas ultimately proposed that pain classifications were unhelpful for purposes of clinical assessment of the causal bases for different types of pain mechanisms. *Id.* at 46, 48–49.

Dr. Aradillas moved on to a review of the categories of conditions that might best describe Petitioner's constellation of symptoms. He noted (in agreement with literature filed in support of this action)[5] that pain deemed "neuropathic" could be the result of a wide variety of diseases—not all of which would *themselves* be considered to be the result of direct neurologic injury. Tr. at 52. CRPS, for example, is a condition that is not readily attributable to an identified structural abnormality, but is often associated with small fiber neuropathy—a condition that is characterized by a measurable diminution in nerve fibers (as revealed by skin biopsy). *Id.* at 49–50. The former would not be deemed classically neuropathic, but the latter would, simply because the association with nerve injury could be more easily made. *Id.* at 51. Some pain syndromes could be considered neuroimmune disorders in and of themselves, however, in which "the immune system is causing these aberrant responses to keep going." *Id.* at 74.

From this, Dr. Ardillas embarked on a lengthy discussion of the physiologic structure and performance of different kinds of nerves in conjunction with the central nervous system ("CNS")

[5] T. Jensen et al., *A New Definition of Neuropathic Pain*, 152 PAIN 2204, 2204 (2017), filed as Ex. 64 on Feb. 28, 2019 (ECF No. 68-10) (noting that "[n]europathic pain is not a single disease, but a syndrome caused by a range of different diseases and lesions, which manifests as an array of symptoms and signs.").

in causing the sensation of pain. *See generally* Tr. at 53–65. He particularly focused on how synapse relay error might produce chronic pain, comparing the situation to a radio whose volume control could not be turned down—a process he termed "sensitization." *Id.* at 60–62. The peripheral version of sensitization occurs at the dorsal root ganglion (located near, and emanating from, the spinal cord). *Id.* at 62, 64–65, 68. In Dr. Aradillas's view, an "out-of-control inflammatory response" in the body (instigated by some other initial injury) could cause T cells central to the immune process and triggered by injury to migrate to the dorsal root, interfering with nerve signal transmission and leading to peripheral nerve sensitization. *Id.* at 70–72.

Dr. Aradillas's specific theory, as he explained at hearing, involves several interlocking components. Tr. at 76. He proposed that "minor tissue damage" (as insignificant as a skin prick from a needle) could in rare circumstances be sufficient to later result in chronic pain. *Id.* at 76–77. This could occur whether the damage specifically involved the nerve or occurred around it—for in either case, the resulting inflammation would have the capacity to cause peripheral sensitization. *Id.* at 78. However, Dr. Aradillas seemed to give greater emphasis to damage to a nerve itself as likely to trigger such a process (although he later also discounted this as the only way by which a pain syndrome could arise). *Id.* at 78–79.

Dr. Aradillas's theory did not assume that the fact of Ms. Todd's being vaccinated was an example of the harm possible from a mere "needle prick," even though he maintained this was a plausible explanation for similar kinds of chronic pain. Tr. at 80, 179–80. Rather, in Ms. Todd's case it was the components of the vaccine itself that explained her subsequent injuries. The flu vaccine was designed to trigger an inflammatory response via both the innate and adaptive immune system, which in his view had a greater capacity to elicit inflammation around nerves than the needle used to administer the vaccine. *Id.* at 80–82, 181.

To describe the mechanistic process by which the aforementioned would theoretically occur, Dr. Aradillas cited several items of literature regarding "autoimmune pain disorders," which he defined as disorders "that manifest[] as pain, as chronic pain, really," driven by autoantibodies. Tr. at 82–84, 89; D. Carr et al., *Neuropathic Pain: The Immune Connection*, 12 Pain: Clinical Updates 1, 2 (2014), filed as Ex. 19 on Feb. 27, 2017 (ECF No. 41-7) ("Carr"). Carr specifically observed that certain antibodies injected into the serum of animal subjects with CRPS would cause the test subjects to develop a version of CRPS—thus demonstrating an autoimmune process. Tr. at 83; Carr, *supra*, at 2.[6]

Another article, in Dr. Aradillas's view, supported the notion that autoantibodies could interfere with nerve function even if they did not harm the actual nerves. Tr. at 84–85; M.

[6] Dr. Aradillas differentiated the precise mechanism by which the proposed antibody cross-reactive attack would occur in such circumstance from the way antibodies might cause other kinds of autoimmune injury, like GBS. Tr. at 83. In some instances, the antibody in question was thought to bind to a self-structure, causing damage, whereas for CRPS with concurrent neuropathic pain, it was more likely that the antibody caused nerve malfunction merely by interfering with the manner in which nerves conduct signals—but without direct harm to the nerve. *Id.* at 83–84, 89–91.

Talkington, *An Autoimmune Basis for Chronic Pain?*, Pain Research Forum 1 (Sep. 4, 2012), filed as Ex. 74 on Feb. 28, 2019 (ECF No. 69-10) ("Talkington"). Talkington discusses an individual who complained of burning pain but revealed no neurologic injury on testing. Talkington, *supra*, at 1. Nevertheless, when the individual was treated with immunotherapies, like intravenous steroids, as if he suffered from an autoimmune condition, he improved. Tr. at 85–86; Talkington, *supra*, at 1–2. Dr. Aradillas himself had confirmed the efficacy of such treatments, like plasmapheresis, in his own patients suffering from CRPS (with those experiencing small fiber neuropathy doing even better). Tr. at 86–87. However, Dr. Aradillas later admitted that he was not proposing that Ms. Todd in fact possessed any autoantibodies that would arguably interfere with her nervous system signal processing sufficient to cause chronic pain—or that this entire line of testimony accurately captured her particular circumstances. *Id.* at 91. Rather, he raised the concept only to underscore the existence of reliable science supporting the notion that a pain syndrome did not have to involve direct damage to the nerves. *Id*. at 92–93.[7]

Dr. Aradillas's testimony said little directly relevant to the nexus between the flu vaccine and the aforementioned processes—a point underscored during his cross-examination. He admitted, for example, that he could identify no direct evidence setting forth how components of the flu vaccine (which are changed on a yearly basis, in anticipation of what wild version of the virus is anticipated to be prevalent) could produce the kind of autoantibodies he purported were interfering or damaging nerve function sufficient to cause Petitioner's symptoms, relying instead on the general assertion "that's how a vaccine works." Tr. at 155–56.

Pivoting from the above, Dr. Aradillas returned to a discussion of how he viewed Ms. Todd's situation. He characterized the pain syndromes associated with peripheral nerve diseases, from GBS to other more general polyneuropathies, as falling under the umbrella of "somatosensory pain system," which he deemed as responsible for transmitting pain signals "in the form of peripheral sensitization." Tr. at 100–02. Peripheral nerve diseases would inherently involve the dorsal root ganglion (which as Dr. Aradillas previously testified was involved in peripheral sensitization). *Id.* at 102. An injury like a small fiber neuropathy would also fit within this structure, and therefore have the capacity to involve a pain disorder. *Id.* at 103–04.

Dr. Aradillas firmly opined that Ms. Todd suffers from some form of small fiber neuropathy (specifically NLDSFN, attributable in this case to an antibody-driven ganglionopathy). Tr. at 120, 133. He classified small fiber neuropathy as a kind of sensory peripheral neuropathy with many possible different presentations, but which would likely implicate interference at the dorsal root ganglion level. *Id.* at 103–04, 106. Small fiber neuropathies result in both "positive"

[7] In the course of this testimony, Dr. Aradillas specifically referenced some articles establishing that the CASPR 1 and 2 antibodies have also been recognized as having the capacity to cause the nerve signal conducting interference in individuals experiencing chronic pain in association with known autoimmune diseases GBS. Tr. at 88–95. But because Dr. Aradillas acknowledged that he does not contend Ms. Todd has ever been shown to possess such autoantibodies, and given that none of her treaters ever proposed testing for their presence, I do not include herein a detailed review of these items of literature. *Id.* at 177–79.

symptoms (meaning affirmative feelings of painful or unpleasant burning or tingling), and "negative" symptoms (characterized by numbness and/or the absence of sensitivity to pressure, heat, and other stimuli), as well as allodynia (pain sensitivity even in the absence of a pain stimulus)[8] but not impairment of motor function. *Id.* at 110–11, 113, 120. A symptom of weakness could be understood as a negative symptom, but could also (if it stemmed from motor difficulties) reflect an entirely different neuropathy. *Id.* at 111.

One distinction in classification of small fiber neuropathies delineated by Dr. Aradillas, however, involved length-dependent (meaning one that "starts at the feet" and ascends into the body for a more widespread impact) versus non-length-dependent (one that is more localized to the ganglion level or impacting only a limited area of the body). Tr. at 107–08, 113–14, 117; Petitioner's Glossary of Terms, filed as Ex. 87 on Feb. 28, 2019 (ECF No. 71-3). As he explained, NLDSFN is less common than its counterpart, and is characterized by a patchy distribution of symptoms that can vary more widely than in the length-dependent version, and can affect a nerve anywhere from the root ganglion to a receptor ending at the skin level. Tr. at 113–14, 116–17; S. Khan et al., *Characterization of Non-Length-Dependent Small-Fiber Sensory Neuropathy*, MUSCLE & NERVE 86, 86 (2012), filed as Ex. 58 on Feb. 28, 2019 (ECF No. 68-4) ("Khan"). He thus asserted that there were "several different types" of NLDSFN, with a ganglionopathy constituting merely one version. Tr. at 166.

There are also different diagnostic tests for each. In Dr. Aradillas's view, length-dependent small fiber neuropathies are correlated with a positive skin biopsy testing small nerve fiber density (which reveals the extent of nerve destruction), but such testing has less utility in assessing the existence of NLDSFN. *Id.* at 100, 108.[9] Thus, even though Petitioner's skin biopsy unquestionably produced normal results, Dr. Aradillas maintained that the possibility of damage to Ms. Todd's dorsal root ganglion could not be ruled out. *Id.* at 127.[10] Indeed, Dr. Aradillas admitted that NLDSFN can exist even in the face of an otherwise-normal medical exam, and (perhaps as a result) is often considered a "psychogenic disorder." *Id.* at 114–15. Another useful test is qualitative sensory testing, or "QST," which provides objective evidence of a patient's responses to certain types of stimuli. *Id.* at 127–28. Overall, Dr. Aradillas proposed that a patient's clinical presentation

[8] Allodynia is "pain resulting from a non-noxious stimulus to normal skin." *Dorland's* at 51. Dr. Aradillas acknowledged on cross-examination that allodynia was not unique to small fiber neuropathies. Tr. at 169–70.

[9] Dr. Aradillas later stressed the impossibility of performing the kind of nerve biopsy that might help establish the existence of an NLPSFN, because the source of the injury (the dorsal root ganglion) could not be tested without the risk of permanent harm. Tr. at 125–26.

[10] Dr. Aradillas referenced an item of literature that he maintained further diminished the value of the skin biopsy. Tr. at 128–29, citing A. Truini et al., *Does the Epidermal Nerve Fibre Density Measured by Skin Biopsy in Patients with Peripheral Neuropathies Correlate with Neuropathic Pain?*, 155 PAIN 828, 829 (2014), filed as Ex. 50 on Nov. 30, 2018 (ECF No. 63-4). That article, he proposed, demonstrated a lack of association between the degree of pain a patient with small fiber neuropathy might experience and the degree of nerve destruction measured by the skin biopsy. Tr. at 128–29.

plus either a positive skin biopsy *or* QST was sufficient for a small fiber neuropathy diagnosis (although he maintained a skin biopsy was ultimately "irrelevant" to assessing the existence of NLDSFN). *Id.* at 165, 174.

Dr. Aradillas next discussed his views on a possible etiology for NLDSFN. He deemed small fiber neuropathies generally to be autoimmune in origin, citing a single item of literature in support. Tr. at 121; X. Liu et al., *IVIg for Apparently Autoimmune Small-Fiber Polyneuropathy: First Analysis of Efficacy and Safety*, 11 Therapeutic Advances in Neurological Disorders 1, 2 (2018), filed as Ex. 52 on Nov. 30, 2018 (ECF No. 63-6) ("Liu"). Liu observed that treating 52 patients believed to be suffering from small fiber neuropathies with IVIg[11] caused them to show improvement, thereby providing at least circumstantial evidence of the condition's autoimmune character. Tr. at 122; Liu, *supra*, at 7. In so doing, Liu's authors compared small fiber neuropathy to other peripheral neuropathies, such as GBS, known to be driven by immunologic processes like autoantibody attacks on the myelin sheath of nerves. Tr. at 122–23; Liu, *supra*, at 2. Dr. Aradillas admitted, however, that the specific targets for a comparable attack on the small, unmyelinated or thinly-myelinated nerves relevant to a small fiber neuropathy had not yet been identified (although he attempted to diminish the significance of this fact by stressing how little was still known about GBS and similar demyelinating conditions). *Id.* at 123–24.

Other evidence of small fiber neuropathies likely autoimmune character was established by the degree to which the condition is experienced by GBS patients (whose neuropathies at the outset impact large nerves). Tr. at 130–31; A. Uncini et al., *Sensory Guillain-Barré Syndrome and Related Disorders: An Attempt at Systemization*, MUSCLE & NERVE, 464, 464 (2012), filed as Ex. 54 on Nov. 30, 2018 (ECF No. 63-8). Dr. Aradillas went so far to propose that small fiber neuropathies could reflect a subcategory or variant of more commonly understood peripheral neuropathies like GBS, with the former having more of a sensory orientation but not featuring all of the clinical indicia otherwise associated with GBS. Tr. at 131–32. Thus, even though Ms. Todd herself had not been shown to possess the clinical criteria necessary for a GBS diagnosis (such as proper CSF readings), her NLDSFN could still be understood to constitute a GBS variant. *Id.* at 132–33. He later acknowledged, however, that he could not specify the clinical diagnostic criteria for this proposed variant, although he felt the variant was medically valid. *Id.* at 161–63.

To support his diagnostic opinion, Dr. Aradillas provided his read on Ms. Todd's medical history. He deemed her early October 2012 visit corroborative of NLDSFN, since Petitioner at this time complained of patchy symptoms in different places on her body. Tr. at 134–35. Later that month, Dr. Aradillas argued, there was evidence that Petitioner's symptoms were progressing in a more widespread, bilateral manner, with burning pain and sunburn-like skin sensitivity that he considered reflective of allodynia. *Id.* at 136–37. Her lumbar spine MRI ruled out other

[11] IVIg—or intravenous immunoglobulin—is a common treatment for demyelinating autoimmune disorders such as GBS or chronic inflammatory demyelinating polyneuropathy. Liu, *supra*, at 2. It works by modifying B- and T-cells thereby inhibiting antibody production. *Id.*

explanations for her symptoms, while her subsequent stay at Waccamaw eliminated the possibility of "classic" GBS. *Id.* at 137–39. And a Waccamaw neurologist seemed to allow that some kind of sensory GBS variant/small fiber neuropathy caused by vaccination could explain Petitioner's constellation of symptoms. *Id.* at 140–41; Ex. 7 at 8.

Dr. Aradillas found additional support for his diagnostic conclusions from Ms. Todd's MUSC records. Testing performed at MUSC revealed a lack of responsiveness to sensory stimuli, allodynia, and a few other symptoms—not all of which (like areflexia) correspond to a small fiber neuropathy. Tr. at 141–43. Nevertheless, treaters later began to actively explore the possibility of such a diagnosis (although some time after vaccination). *Id.* at 143–44 (discussing Duke treatment from 2014). The consistency of Ms. Todd's characterization of her ongoing pain and allodynia was telling in his view. *Id.* at 146–47. At the same time, however, Dr. Aradillas downplayed the lack of testing corroboration for an autoimmune condition, along with the negative skin biopsy, as not precluding his NLDSFN diagnosis—pointing out some specific positive inflammation biomarker results (although these kinds of tests were, taken as a whole, overwhelmingly negative). *Id.* at 148–49. He found especially significant the fluctuating nature of some of this inflammation testing, since it was consistent with his view that an autoimmune condition would be expected to follow a variable course. *Id.* at 149. And he deemed the timing of onset of Petitioner's symptoms (12 days post-vaccination) to be biologically reasonable, based on how long he expected it would take for symptoms to begin after the initiation nerve function interference. *Id.* at 151–52; P. Austin & G. Moalem-Taylor, *The Neuro-immune Balance in Neuropathic Pain: Involvement of Inflammatory Immune Cells, Immune-like Glial Cells and* Cytokines, 229 J. of Neuroimmunology 26, 36 (2010), filed as Ex. 44 at on Nov. 11, 2018 (ECF No. 62-8).

On cross, Dr. Aradillas admitted that his embrace of NLDSFN as the proper diagnosis (which he acknowledged none of Petitioner's treaters ever proposed) came only after preparation of his original report. Tr. at 163–64, 172–73. He admitted that some other neuropathic conditions that he deemed falling on the same overall spectrum as Petitioner's symptoms, like CRPS, were not applicable to this case. *Id.* at 161–62. He also noted that Petitioner did not in fact display some of the clinical criteria he had deemed critical to an NLDSFN diagnosis. *Id.* at 171. Thus, Ms. Todd tested negative on a skin biopsy—and although Dr. Aradillas steadfastly maintained that this test only ruled out nerve fiber "destruction," rather than the malfunction he maintained she had experienced, he accepted that some of the literature he had filed actually *contradicted* his assertion that skin biopsies were not useful in establishing NLDSFNs. Tr. at 100, 166–67, 187–88, 190; K. Gorson, et al., *Non-length Dependent Small Fibre Neuropathy/Ganglionopathy*, 79 J. Neurosurgery Psychiatry 163, 163 (2008), filed as Ex. 56 on Feb. 28, 2019 (ECF No. 68-2) (finding that skin biopsies could help establish whether study participants had a non-length-dependent small fiber ganglionopathy).

Dr. Aradillas also agreed that Petitioner had never received QST testing, while proposing that she nevertheless did display some altered sensation testing results (and arguing that the overall record still supported the conclusion that she suffered from *some* kind of neuropathic pain

syndrome). Tr. at 170–73. He reiterated his opinion on cross that a QST test result plus clinical evidence of a neuropathic pain syndrome was enough to support a NLDSFN diagnosis, relying on Gorson for this assertion (and despite demonstration by Respondent that Gorson did *not* mention QST testing as a clinical criteria for the diagnosis). *Id.* at 174–76.

Cross examination revealed other evidentiary deficiencies in Dr. Aradillas's theories. For example, although Dr. Aradillas proposed that a vaccine could cause the kind of neuropathic injury that Ms. Todd allegedly suffered simply as a result of "profound inflammation" generated by the innate immune response, he admitted there was a lack of direct evidence establishing the contention, and that it could likely not be measured by normal medical standards (such as the inflammation biomarker tests administered to Ms. Todd, which had consistently not revealed any alarming post-vaccination inflammation). Tr. at 181–83. Rather, this particular theory was (in Dr. Aradillas's view) corroborated by Petitioner's clinical symptoms, and the fact that she displayed a positive ANA in 2014—almost two years after vaccination. *Id.* at 186. He later, however, proposed that it was more likely Ms. Todd's condition was the product of an adaptive immune response, mediated by antibodies—even though (at the same time) he could not specify the relevant antibodies in her case, and noted that many of the antibodies that arguably were associated with an autoimmune-derived neuropathy did not apply to her. *Id.* at 188–90.

C. *Respondent's Experts*

1. Dr. Eric Lancaster

Dr. Lancaster submitted two reports and testified on Respondent's behalf at hearing. Expert Report of Dr. Lancaster, dated Aug. 11, 2017, filed as Ex. B (ECF No. 51-4) ("Lancaster Rep."); Supplemental Expert Report of Dr. Lancaster, dated Feb. 21, 2019, filed as Ex. D (ECF No. 66-1) ("Lancaster Supp. Rep."); Tr. at 198–334. Dr. Lancaster rejected Petitioner's contention that she suffered from NLDSFN, as well as the broader contention that her symptoms were attributable to a vaccine-caused, antibody-driven autoimmune process.

Dr. Lancaster is a clinical physician at the Center for Autoimmune Neurology at the University of Pennsylvania, as well as an assistant professor of neurology at the University of Pennsylvania. Dr. Eric Lancaster Updated CV, filed as Ex. E on March 3, 2019 (ECF No. 72-1) ("Lancaster CV"). He completed a neurology residency at the University of Pennsylvania from 2004–07, and is board certified in neurology, with subspecialties in neuromuscular medicine and electrodiagnostic medicine. *Id.*; Tr. at 199. His research focuses on antibody-mediated neurological disorders, and he sees patients with complex autoimmune neurologic disorders on a regular basis. Lancaster CV at 1; Tr. at 202–04. He has significant expertise performing the tests used to evaluate peripheral neuropathies (e.g., EMGs, nerve conduction studies)[12], and also has a

[12] An EMG, or electromyography test, is a diagnostic procedure used to assess the health of muscles and the nerve cells that control them (motor neurons). *Dorland's* at 602. Nerve conduction studies are used in conjunction with EMGs to detect and locate peripheral nerve injuries or disease. *Mosby's* at 514.

clinical practice. Tr. at 200–02; Lancaster Rep. at 1. Dr. Lancaster has treated patients with small fiber neuropathies. Tr. at 202.

Dr. Lancaster began with an overview of small fiber neuropathies. The term "neuropathy" refers to damage to the peripheral nerve fibers. Tr. at 208. While large nerve fibers are mostly associated with motor function and control in the human body (although they also play a role in detecting position and some other sensations, like vibration), the small fibers (which are either unmyelinated or thinly myelinated)[13] generally "detect pain, cold, heat, and other modalities," such as injuries. Tr. at 208. Although many neuropathies will impact both large and small fibers, small fiber neuropathies are "exclusively" relevant to the small fibers and the functions they serve, preserving the function of the large fibers. *Id.* at 210–11. Dr. Lancaster largely accepted Dr. Aradillas's contentions about the kinds of symptoms a person with a small fiber neuropathy would experience (e.g., burning pain, tingling, loss of sensation/numbness, etc.). *Id.* at 327–28.

"Length-dependent" small fiber neuropathies, in which "the longest fibers get affected first and most predominantly," were in Dr. Lancaster's estimation most common. Tr. at 208–09. In such cases (perhaps due in part to the metabolic strain placed on longer nerve fibers), symptoms ascend from distal portions of the body, like the feet and hands, and move toward the chest (hence closer to the spine and brain stem, where nerve signals would be processed). *Id.* at 209. Length-dependent small fiber neuropathies were more likely to occur than NLDSFNs. 209–11.

A NLDSFN, by contrast, is "a much more variable category." Tr. at 210, 213–14. Dr. Lancaster did not accept (although he did not completely rule out) the possibility that an NLDSFN could be caused by a virus, but he directly disputed that NLDSFNs were properly considered "in general" autoimmune conditions.[14] He acknowledged there existed hypotheses that an NLDSFN could have an autoimmune etiology in some cases, but noted the existence of many *more* recognized etiologies that were not, including metabolic dysfunction, diabetes, and other illnesses. *Id.* at 215–16. He also deemed significant the total number of individuals with neuropathies, which he placed in the millions, that were considered either attributable to diabetes or an idiopathic source. *Id.* at 216. Because of the usual etiologies for NLDSFN, the condition is most effectively addressed with treatments aimed at the underlying cause, such as control of blood sugar where diabetes is at issue, or vitamins to treat underlying vitamin deficiencies believed to be causing the

[13] Myelination is the process by which the body produces a fatty substance that coils around nerve cells providing a protective sheath and electrical insulator. *Dorland's* at 1218.

[14] Dr. Lancaster also questioned whether there existed definitive evidence establishing a particular autoantibody as capable of causing NLDSFN without other neurologic symptoms, although he allowed it might be established someday. Tr. at 234. He also emphasized (based upon his experience studying antibody-mediated neuropathies) that the kind of syndromes where a particular antibody, like CASPR2, is thought to result in *some* symptoms comparable to a NLDSFN involve a broader and more complex presentation than what Petitioner claims to have experienced. *Id.* at 283–95, 297–304.

neuropathy. *Id.* at 230–32. Where the source for the neuropathy was deemed idiopathic, by contrast, the best course was to attempt to reduce pain symptoms, but not with immune therapies. *Id.* at 233.

The NLDSFN subtype most relevant herein is a ganglionopathy, in which "the cell body itself [of the dorsal root ganglia] is injured in the ganglia root[15] and dies, or becomes impaired or defective in some way," independent of the length of the targeted nerve. *Id.* at 211, 214. This results in symptoms presenting as changes in sensation sensitivity occurring in a "non-length-dependent pattern." *Id.* Like Dr. Aradillas, Dr. Lancaster agreed that such changes could result in positive as well as negative phenomena, combining a loss of feeling sensitivity to various stimuli with abnormal pain elsewhere plus allodynia, all caused by neuron-signaling malfunction. *Id.* at 215, 217–18, 221. Dr. Lancaster proposed that there was no functional difference between the terms NLDSFN, "non-length-dependent ganglionopathy," and "dorsal root ganglionopathy," since all three described a process exclusively affecting small fiber nerves, with an unproven "presumption" that the ganglia are the target. *Id.* at 212–13. However, existing literature in Dr. Lancaster's view (including what Petitioner offered) did not reliably demonstrate that "the ganglia is actually the site of any particular pathology," and that what research that did support the concept involved "excruciatingly rare" diseases. *Id.* at 212.

In describing the clinical or testing indicia relevant to diagnosing NLDSFN, Dr. Lancaster reviewed several items of literature, including Gorson. Tr. at 213–30. Most individuals suffering from NLDSFN would likely experience persistent symptoms in the same area—consistent with the loss of nerve fibers in the presenting area of the body, causing "fixed deficits" reflecting the destruction of the nerve cells. *Id.* at 224–25. Patients would also likely have a loss of pin prick sensation in the presenting areas. *Id.* at 229. Reflexes, since they pertain to motor nerve function, would not bear on a NLDSFN diagnosis. *Id.* at 269–71.

With respect to testing, Dr. Lancaster maintained that one article filed by Petitioner very plainly endorsed the skin biopsy test[16] for diagnosing NLDSFN. Tr. at 221–23, 229–30; *see generally* Khan, *supra* (discussing study design that only included patients with a skin biopsy diagnosis of NLDSFN).[17] Also, and contrary to Dr. Aradillas, he argued that direct biopsy of the

[15] Although Dr. Aradillas spoke of nerve "dysfunction" as possible even without direct nerve damage, Dr. Lancaster expressed skepticism that this was the most common accurate explanation for SFN, deeming nerve malfunction absent damage a "super rare phenomenon." Tr. at 333. In the majority of SFN cases, actual nerve damage (as reflected in skin biopsies) was the more likely culprit. *Id.* at 333–34.

[16] In his testimony, Dr. Lancaster described how a skin biopsy testing for SFN is performed, and what it captures. Tr. at 223–24.

[17] In response to cross-examination questioning, Dr. Lancaster disputed Petitioner's contention that Khan in fact did not completely establish the efficacy of the skin biopsy test, given that a small percentage of tested individuals did not display abnormal results. Tr. at 317–18. Dr. Lancaster proposed that the proper reading of this part of Khan was that

dorsal root ganglion was possible, although he admitted that it was generally not done due to the burdensome nature of such testing. Tr. at 311–12. Nevertheless, the skin biopsy test was still valuable (and more easily performed), even if it was somewhat indirect, and even if it was not 100 percent predictive in all cases. *Id.* at 311, 319–20. QST, testing, by contrast, was not in Dr. Lancaster's opinion useful for diagnosing NLDSFN. Tr. at 226. While he allowed that QST testing had some value, it largely overlapped a neurologist's own examination of a patient, was more illustrative of symptoms, and overall had "lower specificity" and "lower sensitivity" than a biopsy, which provided objective evidence of nerve injury. *Id.* at 226–28. He also noted that literature like Khan reflected the lower significance given to QST testing in comparison to the biopsy. *Id.* at 228.

Dr. Lancaster relied on several record facts for concluding that Ms. Todd likely did not have NLDSFN. He granted that her symptoms "at least made it something reasonable to think about." Tr. at 230. But he found more compelling the objective evidence—the lack of a positive skin biopsy, as well as inconsistency in her "areas of confirmed loss of sensation"—pointing in the opposite direction. *Id.* He characterized her overall presentation (which coupled neuropathic-like symptoms without other neurologic complaints) as "quite a common phenomena" in his experience, but attributing the cause of symptoms in such cases to an unidentified idiopathic source. *Id.* at 235.

To support his opinion, Dr. Lancaster provided a detailed review of Petitioner's medical history. *See generally* Tr. at 235–83. He noted first that the late-October 2012 record set forth symptoms that in his view were not consistent with a small fiber neuropathy, such as decreased sensation of a pin prick or temperature "in a particular distribution," and thus the relevant treater (Dr. Triana) did not propose any kind of small fiber neuropathy as explanatory. Tr. at 238. The neurologist Petitioner subsequently saw at Waccamaw, Dr. Amodeo, "documented a pretty detailed neurologic examination" that did not reveal meaningful weakness, but did establish some touch sensitivity, and although he allowed for the possibility of an autoimmune peripheral neuropathy, like GBS, or a dorsal root ganglionopathy (which would be closer to what Petitioner alleges she experienced), he could not provide a certain diagnosis (and did not otherwise propose NLDSFN). *Id.* at 243–45, 247. Dr. Lancaster, however, stressed that his personal view (based on consideration of such records) was that Ms. Todd's sensory symptoms at that time were by themselves insufficient to support a NLDSFN diagnosis. *Id.* at 245–46.

At MUSC, Dr. Lancaster noted, Petitioner again received a neurologic exam from Dr. Pritchard that came back largely normal, although she was suffering from sufficient pain to interfere with the exam's completion. Tr. at 247–51. However, even under such circumstances Dr. Lancaster felt the exam did not suggest "definitive" weakness. *Id.* at 252–53. He also felt that the evidence of loss of sensation at this time was insufficient to establish the existence of a neuropathy,

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a small group of patients did not display nerve density abnormality in *both* tested sites (proximal and distal thigh), but that *all* showed abnormality in at least one tested location consistent with NLDSFN. *Id.* at 319.

and this plus the lack of other "objective deficits," (including close-to-normal reflexes) reasonably led treaters to be inconclusive in their assessment of Petitioner's condition. *Id.* at 254–55, 257–58. At a minimum, however, Dr. Lancaster felt the results of this examination did *not* support a NLDSFN diagnosis, or any other neuropathy. *Id.* at 255. Petitioner's discharge confirmed the fact that even taking into account her touch sensitivity, treaters "did not find those sort[s] of sensory deficits that you would want to diagnose neuropathy or another neurologic disorder." *Id.* at 261.

Nearly a year later, Dr. Lancaster observed, Ms. Todd returned to Dr. Pritchard at MUSC in June 2013. *See generally* Tr. at 262–64; Ex. 82 at 6. At this time, she reported some flareups of her skin sensations, tingling, plus a skin eruption that raised concerns about a dermatomal distribution that could establish shingles. *Id.* at 263.[18] However, her exam was again largely normal, and Dr. Pitchard was unable to offer a diagnosis. *Id.* at 264. Then, at a later visit in September 2013 with a neuromuscular neurologist (a specialist Dr. Lancaster deemed qualified to evaluate the existence of small fiber neuropathies), another comprehensive workup established insufficient objective evidence of neuropathy (although treaters seemed willing to explore the matter further). *Id.* at 265–68; Ex. 10 at 12–15. Notably, Petitioner reported symptoms prior to this examination but was asymptomatic on exam—something that Dr. Lancaster deemed inconsistent with NLDSFN, which we felt (as previously noted) would involve persistence of at least sensory deficits rather than variable "acute attacks" of pain followed by lulls. Tr. at 268.

Dr. Lancaster observed the same lack of support for the NLDSFN diagnosis in records from 2014. Tr. at 272–83. Petitioner's examination in the spring of 2014 from Dr. Hartsell at Duke did not produce evidence of neuropathy. *Id.* at 274–76. And the skin biopsy test Dr. Hartsell ordered produced normal density results. *Id.* at 275–77. Dr. Lancaster's own reading of the test results, as set forth in the medical record, led him to opine that they were unsupportive of a NLDSFN diagnosis. *Id.* at 278–79. Petitioner's subsequent follow-up visit with Dr. Hartsell that fall resulted in his identifying fibromyalgia (and not NLDSFN) as a working diagnosis, something Dr. Lancaster felt was a generic diagnostic determination in the absence of any more specific, neuropathic explanation. *Id.* at 280–83.[19]

Besides addressing the nature of Petitioner's injury and its purported autoimmune cause, Dr. Lancaster's testimony also covered some of the other core elements of any Vaccine Program

[18] Dr. Lancaster explained that the varicella virus that causes shingles can be dormant in the dorsal root ganglia but, upon reactivation, will follow a pattern in the skin "supplied by the sensory fibers in the dorsal root ganglia" resulting in a blistering rash. Tr. at 263.

[19] Dr. Lancaster did note that although the records from Petitioner's November 2014 visit to Dr. Hartsell suggested the possibility that she has "overlapping post-AIDP [acute inflammatory demyelinating polyneuropathy] neuralgia," he did not find support in the record that in fact she was ever previously diagnosed with AIDP, which is essentially "the most common form of GBS." Tr. at 279–80. I similarly find no evidence that this note reference has any pre-existing evidentiary basis in the record, and would not be able to conclude that Petitioner's symptoms were attributable to GBS even had she so alleged.

claim. He thus disputed directly whether the flu vaccine could cause any form of small fiber neuropathy (and did not find persuasive case reports filed by Petitioner that purportedly established the contrary). Tr. at 234, 329–30. Consistent with Dr. Aradillas's admission, he observed that not only was there no evidence that Petitioner possessed any of the antibodies that could potentially cause a neuropathic syndrome featuring some of the symptoms Petitioner experienced, but they could easily have been tested for—something no treater who saw Ms. Todd ever proposed (and he added that he would not have either had he treated her). *Id.* at 295–96, 307–08. He did not believe the record supported the conclusion that Ms. Todd otherwise had such a syndrome. *Id.* at 307–09. And he maintained that he was not aware of reliable evidence establishing the capacity of the flu vaccine to even cause the production of such antibodies. *Id.* at 296–97.

On cross-examination, Dr. Lancaster admitted that some autoimmune conditions could possibly involve viral-caused flares. Tr. at 315. But he did not accept the proposition that the possession of one kind of autoimmune biomarker, such as a positive ANA, invariably led to the conclusion that the individual in question had an autoimmune condition. *Id.* at 325. Similarly, he discounted the evidentiary value of IVIg treatment as indirectly corroborating that a person likely had an underlying autoimmune illness, noting that such treatments had a "massive placebo effect," making it difficult to conclude merely from a patient's reported improvement, and absent some other objective corroboration (like a reduction in suspected antibodies believed to be causing the condition) that the treatment was likely addressing the true cause of her symptoms. *Id.* at 326–27.

In addition, Dr. Lancaster accepted the possibility of vaccine-caused adverse events, such as GBS caused by the flu vaccine. Tr. at 321–23. He also agreed that the medical records established that Ms. Todd had repeatedly complained of burning pain. *Id.* at 329. He disagreed, however, with the statement of Ms. Todd's primary care physician, Dr. Crosby, that she should not in the future receive the flu vaccine. *Id.* at 324–25.

2. Dr. Penelope Morel

Dr. Morel, a research and teaching immunologist, served as Respondent's second expert, filing two reports and testifying at hearing. First Report of Dr. Morel, dated Aug. 11, 2017, filed as Ex. A (ECF No. 49-1) ("Morel First Rep."); Second Report of Dr. Morel, dated Feb. 18, 2019, filed as Ex. C (ECF No. 65-1); Tr. at 335–380. She offered the opinion that the flu vaccine Ms. Todd received did not play a role in the neurologic symptoms she subsequently experienced. Tr. at 342.

Dr. Morel is currently employed by the University of Pittsburgh School of Medicine Department of Immunology and the University of Pittsburgh Cancer Institute. Dr. Penelope Morel CV, filed as Ex. A Tab 1 on Aug. 14, 2017 (ECF No. 49-2) ("Morel CV"). She also serves as an affiliate member at the University of Pittsburgh Center for Vaccine Research. Morel CV at 2. Dr. Morel obtained a Bachelor of Medicine degree from the University of South Hampton in the

United Kingdom. *Id.* at 1. She then obtained a Doctor of Medicine degree from the University of Geneva in Switzerland while performing research in the World Health Organization Immunology and Training Lab. Morel CV at 1; Tr. at 336. She later completed a postdoctoral fellowship at Scripps Clinic and Research Foundation in La Jolla, California as well as a fellowship in immunology at Stanford University Medical Center in Stanford, California. Morel CV at 2. She completed a second postdoctoral fellowship in molecular genetics at the University of Pittsburgh Cancer Institute. *Id.*

Dr. Morel does not have a clinical practice, and instead focuses on research. Tr. at 363–64. She has published numerous articles on immunological responses to vaccination, and she has specifically focused on immune response regulation, autoimmune diseases and the genetic predisposition to such diseases, and the immunologic mechanisms that prevent them. Tr. at 338–41. While Dr. Morel indicated that she was familiar with immunological responses to vaccination, including inflammation, she does not consider herself to be an expert on pain medicine and has not dedicated her research to the topic. *Id.* at 371. She also indicated that she would defer to the opinions of Dr. Lancaster and Petitioner's treating physicians regarding Petitioner's proper diagnosis. *Id.* at 342.

Although Dr. Morel did not offer an opinion as to the proper diagnostic understanding of Petitioner's symptoms, she commented on the association between those kinds of symptoms and what she knows about autoimmunity in general. Thus, she emphasized that not all chronic neuropathic pain can be deemed autoimmune in nature. Tr. at 343. Where autoimmunity *was* a likely cause, testing would reveal "evidence of an aberrant immune response," such as the presence of biomarkers or test results associated with systemic inflammation. *Id.*; First Morel Rep. at 3. She allowed that an immune-associated neuropathic condition would prove responsive to "immune-modulating therapies." Tr. at 354; First Morel Rep. at 3.

Turning to the record in this case, Dr. Morel noted the extent to which various testing results did not suggest Ms. Todd was in fact experiencing the kind of inflammation that would be expected in an autoimmune-driven pathologic process. For example, hematology results from testing performed at the end of October 2012 (five weeks after the vaccination in question) revealed a normal ESR—a well-recognized, albeit nonspecific, test for the presence of systemic inflammation occurring in the context of an autoimmune process. Tr. at 344–45, 351. The CRP test (another kind of inflammation test) was also unsupportive of active inflammation. *Id.* at 345. Dr. Morel would have expected elevated readings if in fact Petitioner had at this time been suffering from an autoimmune process. *Id.*

Testing performed in early November 2012 (when Petitioner was at Waccamaw) for certain autoantibodies associated with inflammatory vascular illnesses also revealed normal results, as well as several other known autoimmune conditions. Tr. at 346–47. Then, additional testing performed almost two years later yielded results consistent with what had been seen more contemporaneously with Ms. Todd's vaccination. Tr. at 350. Thus, inflammation testing continued

to produce normal readings[20] and Petitioner tested negative for Lyme disease antibodies. *Id.* at 348. Petitioner did display a low positive ANA, but Dr. Morel considered this nonspecific (especially since a meaningful number of otherwise-healthy individuals can test positive for this antibody) and therefore not necessarily "clinically relevant." *Id.*; *see also* Tr. at 364–65.[21] The result nevertheless led Petitioner's treaters to propose a rheumatologic consult in August 2014, but (as the records reflect) she was ultimately not found to be likely suffering from any such injury despite her ongoing symptoms. *Id.* at 349–50.

Besides serologic testing, Dr. Morel also found significant Petitioner's nerve biopsy results from her spring 2014 workup at Duke. She noted that the results did not reveal vasculitis or any other "histological abnormalities." Tr. at 352–53; Ex. 88 at 14. In addition, the skin biopsy performed on May 9, 2014 was only consistent with immune infiltrates that would be associated with a localized rash, as opposed to the broader neurologic/sensory symptoms of which Petitioner primarily complains. Tr. at 353, 366–67.

Another way of understanding the lack of support for the contention that Petitioner's condition was autoimmune-derived, Dr. Morel reasoned, was how Petitioner's physicians approached treatment and testing generally. Ms. Todd was never tested for the autoantibodies known to be associated with neurologic diseases—something Dr. Morel felt would have occurred if Petitioner's treaters had even entertained the possibility that her symptoms were autoimmune in origin. *Id.* at 352. In addition, treaters never proposed that she receive any immune-modulating therapies. *Id.* at 354. She did, however, admit that Petitioner had reported some immediate improvement after such treatment, although she would need to see sustained improvement if she was going to accept that the treatment revealed the autoimmune nature of Petitioner's claimed injury. *Id.* a 368–69.

Besides the above, Dr. Morel commented, from her vantage as an immunologist, on certain aspects of Dr. Aradillas's opinion. She noted one article Petitioner filed that seemed to associate high-positive ANA results plus evidence of positive CRP testing with individuals suffering from small fiber neuropathies. Tr. at 351; M. Lang et al., *Diagnostic Value of Blood Tests for Occult Causes of Initially Idiopathic Small-Fiber Polyneuropathy*, 263 J. of Neurology 1, 6 (2016), filed as Ex. 86 on Feb. 28, 2019 (ECF No. 71-2)("Lang"). In this case, however, not only was Petitioner's positive ANA (measured in 2014) much lower than what Lang proposed, but Petitioner herself had never been unequivocally diagnosed with a small fiber neuropathy—unlike Lang's subjects. Tr. at 351–52; Lang at 3. She also emphasized her overall view (consistent with her

[20] Dr. Morel acknowledged that Petitioner did display a slightly elevated CRP reading in August 2014, but she deemed it marginal since it was barely over the high end of the normal range. Tr. at 350. She also noted (along with the positive ANA) that these findings occurred were two years after vaccination—too long to deem likely associated. *Id.* at 355.

[21] On cross, Dr. Morel noted that she did not find significant the fact that Ms. Todd had initially had a negative ANA, followed by a positive one later, given the lack of corroborative evidence of systemic inflammation from other testing. Tr. at 365. She also noted that a person with a rheumatic condition would possess a positive ANA *plus* other evidence specific to conditions. *Id.* at 366.

reading of the medical record) that, contrary to certain assertions in Dr. Aradillas's report, there was a lack of evidence (such as proof of nonspecific inflammation) suggesting that Ms. Todd's receipt of the flu vaccine resulted in an initial aberrant innate immune response. *Id.* at 361–62.

III. Procedural History

After the case's initiation in August 2015, medical records were filed until the spring of 2016. Thereafter, the parties began obtaining and filing their expert reports (as referenced above), completing the process by January 2018. In the interim period, the case was reassigned to me, and after a status conference with the parties I set the matter down for a hearing to be held in March 2019. *See* Prehearing Order, dated June 1, 2018 (ECF No. 59). The hearing occurred as scheduled, with the parties filing simultaneous post-trial briefs in early July 2019. The matter is now ripe for resolution.

IV. Relevant Law

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 11(c)(1), 13(a)(1)(A), 14(a); *see also Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[22] In this case, Petitioner does not assert a Table claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(a)(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enters. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions;

[22] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. App'x 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health & Human Services*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Human Servs.*, 121 Fed. Cl. 230, 245 (2015), *vacated on other grounds*, 844 F.3d 1363 (Fed. Cir. 2017).

In discussing the evidentiary standard applicable to the first *Althen* prong, many decisions of the Court of Federal Claims and Federal Circuit have emphasized that petitioners need only establish a causation theory's biological plausibility (and thus need not do so with preponderant proof). *Tarsell v. United States,* 133 Fed. Cl. 782, 792–93 (2017) (special master committed legal error by requiring petitioner to establish first *Althen* prong by preponderance; that standard applied only to second prong and petitioner's overall burden); *Contreras,* 121 Fed. Cl. at 245 ("Plausibility . . . in many cases *may* be enough to satisfy *Althen* prong one." (emphasis in original)); *see also Andreu*, 569 F.3d at 1375. At the same time, there is contrary authority from the Federal Circuit suggesting that the same preponderance standard used overall in evaluating a claimant's success in a Vaccine Act claim is also applied specifically to the first *Althen* prong. *See, e.g.*, *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010) (affirming special

master's determination that expert "had not provided a 'reliable medical or scientific explanation' *sufficient to prove by a preponderance of the evidence a medical theory* linking the [relevant vaccine to relevant injury]."") (emphasis added). Regardless, one thing remains: petitioners always have the ultimate burden of establishing their Vaccine Act claim *overall* with preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell*, 133 Fed. Cl. at 793 (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *see also Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting *Althen*, 418 F.3d at 1280)). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and/or statements of a treating physician's views, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted."). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (finding that it is not arbitrary or capricious for special masters to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without op.*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant

proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.*; *see also Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Analysis of Fact Evidence*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Human Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *see also Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("Given the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"); *Rickett v. Sec'y of Health & Human Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Human Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at

1525 (Fed. Cir. 1993) ("[I]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms.").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs*., No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Human Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. denied sub. nom. Murphy v. Sullivan*, 506 U.S. 974 (1992) ("It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.") (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1948)).

There are, however, situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Human Servs*., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); *Lowrie*, 2005 WL 6117475, at *19 ("Written records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent.") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Human Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Human Servs*., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Human Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). But nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs*., No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 Fed. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters

must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("Assessments as to the reliability of expert testimony often turn on credibility determinations…."); *see also Porter v. Sec'y of Health & Human Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("[T]his court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act.").

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not every filed item factors into the outcome of this decision. While I have reviewed all of the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *See Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Human Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. The Record Does Not Support the Conclusion that Petitioner Had an NLDSFN

In many Vaccine Act cases, determining whether the evidence supports the injury alleged by the petitioner is a precursor to (and can even supplant) evaluation of the petitioner's success in satisfying the *Althen* prongs. *LaPierre v. Sec'y of Health & Human Servs.*, No. 17-227V, 2019 WL 6490730, at *16–17 (Fed. Cl. Spec. Mstr. Oct. 18, 2019) (citing *Broekelschen*, 618 F.3d at 1346. This is especially critical where the claimant's theory of causation relies on a fact finding for the alleged injury, i.e. where the theory articulates a vaccine-initiated process that invariably leads to a particular kind of injury, and/or where it is accepted by science and medicine that a particular injury results from an immunologic process associated with the vaccine at issue. *Lapierre*, 2019 WL 6490730, at *17. This is the case herein—not only did Dr. Aradillas plainly embrace NLDSFN as the best diagnostic descriptor for Ms. Todd's constellation of symptoms, but the scientific and medical theories he proposed for how the flu vaccine could have brought about this injury were fairly specific to this kind of small fiber neuropathy.

Here, however, the record and expert testimony taken together do not support the conclusion that Petitioner suffered from NLDSFN. Critically, *no* treater ever proposed that Petitioner had such a neuropathy. Nor does the record suggest the existence of any of the indicia that would bulwark Petitioner's diagnostic contention. Numerous test results—especially those performed close in time to Ms. Todd's receipt of the flu vaccine—do not establish the existence

of the sort of systematic inflammation that would be associated with an autoimmune neuropathy. Petitioner never tested positive for any autoantibodies that could even *possibly* be linked to a small fiber neuropathy. And Respondent's experts persuasively established that a positive ANA finding (almost two years post-vaccination, moreover) is too nonspecific to constitute even indirect evidence of a small fiber neuropathy. Dr. Lancaster also persuasively established that the inconsistent, waxing and waning nature of Petitioner's symptomatic complaints for him was another factor ruling out NLDSFN as the proper diagnosis. Tr. at 224–25. Finally, the evidence in this case strongly supports the conclusion that a skin biopsy *is* in fact the most accurate test for small fiber neuropathies. Khan, *supra*, at 89. But Petitioner's test results were negative. Ex. 88 at 14.[23]

Dr. Aradillas could not overcome what the record plainly shows with his alternative view of the proper diagnosis. First, his expertise is mainly in the treatment of pain syndromes and disorders, as opposed to the clinical assessment of neuropathic injury—so, although he had sufficient expertise and background in neurology to offer an opinion in this case, he did not have the *specific* grounding in neuropathies generally to offer a *persuasive* opinion. Second, his opinion tried unsuccessfully to navigate around inconvenient facts in the record—for example, by arguing with limited reliable scientific support) that even though Ms. Todd's record does not establish the kind of actual nerve injury that would be associated with an autoimmune neuropathy, nerve "malfunction" can also explain NLDSFN. This contention, while not implausible, was not demonstrated to establish what most commonly causes NLDSFN, let alone that this characterized Ms. Todd's condition. Dr. Aradillas also was not credible in suggesting other proper diagnostic and clinical criteria for the condition.

Dr. Lancaster, by contrast, more persuasively undermined Petitioner's injury contentions. He noted that Petitioner's negative skin biopsy as well as the inconsistency in her "areas of confirmed loss of sensation" were not indicative of NLDSFN. Tr. at 230. He also noted that many of the physicians who treated Petitioner documented normal or insignificant examination findings. Tr. at 238, 243–45, 247–51, 274–77. And he, along with Dr. Morel, showed how the overall record did not support the conclusion that Petitioner was suffering from the kind of persistent inflammation that would be congruent with the conclusion that she had an autoimmune-caused illness (which Dr. Aradillas largely seemed to contend characterized NLDSFN). Though Dr. Lancaster allowed that NLDSFN was not an unreasonable proposal under the circumstances, given the nature of some of Petitioner's symptoms, he ultimately concluded that it was not supported by the record, and I give greater weight to his testimony and opinion in this case.

[23] By contrast, the literature and more persuasive expert testimony does not support Dr. Aradillas's contention that a positive QST test could support an NLDSFN diagnosis—and in any event, Petitioner does not appear to have ever received such a test.

II. Petitioner Has Not Satisfied the *Althen* Test for Entitlement

As noted, Petitioner's case depended on a finding that she experienced NLDSFN. I find instead that the record evidence only preponderates in the determination that her symptoms had an idiopathic origin—thus obviating the need for a complete *Althen* analysis, since Petitioner's theory required that I find the existence of a specific injury. Nevertheless, even if my determination had been otherwise, or I had found that Petitioner suffered from some kind of generalized neuropathy arguably autoimmune in origin, I would still be unable to rule that Petitioner carried her overall burden of proof under *Althen*.

First, Petitioner's showing on the "can cause" *Althen* prong was insufficient. The best understanding of the theory espoused by Petitioner is that the flu vaccine could cause general interference with nerve function (as opposed to direct injury to the small fibers, given that Dr. Aradillas seemed to concede there is no evidence in this case that this occurred), via an adaptive immune response to the flu vaccine attributable to the production of autoantibodies. Some literature support was offered detailing other contexts in which autoantibodies are thought to interfere with *other* biologic processes, such as when the patients experiencing GBS produce antibodies against the CASPR1 antigen resulting in subsequent pain. Tr. at 90–93; A. Goebel, *Autoantibody Pain*, 15 Autoimmunity Reviews 552, 555 (2016), filed as Ex. 62 on Feb. 28, 2018 (ECF No. 68-8).

But numerous links of the theory as applicable in *this specific case* were wanting or nonexistent. Thus, little reliable evidence was offered showing that (a) the flu vaccine has been shown to cause the creation of antibodies of the sort capable of interfering with nerve function, or (b) that this kind of interference has been offered to explain NLDSFN, as opposed to the kind of direct, demyelinating harm known to be implicated in other peripheral neuropathies, like GBS (which of course it is undisputed Petitioner did *not* have). And Dr. Aradillas was at the limit of his expertise in opining how a flu vaccine could produce a NLDSFN, further reducing the effectiveness of his explanations. Dr. Lancaster, by contrast, was effective and persuasive in establishing that NLDSFN was more likely than not in most cases the product of the kind of direct nerve damage that Petitioner concedes is absent here, and that it is otherwise not properly considered an autoimmune-caused injury, at least directly.

I acknowledge, however, that other decisions have found that a vaccine can precipitate a small fiber neuropathy—albeit via the kind of direct, demyelinating injury that is plainly not demonstrated by the record in this case. *See, e.g.*, *Swaiss v. Sec'y of Health & Human Servs.*, No. 15-286V, 2019 WL 6520791 (Fed. Cl. Spec. Mstr. Nov. 4, 2019) (finding that a petitioner claiming small fiber neuropathy following receipt of the tetanus-diphtheria-acellular pertussis vaccine was entitled to compensation). Accordingly, and despite the overall unpersuasive showing on the first *Althen* prong made herein, there does exist some reasoned support for the theory.

But even if I had simply adopted the reasoning from such other cases, and found that Petitioner preponderantly established the first *Althen* prong, Petitioner's case would then run aground on the second, "did cause" prong. For the record does not confirm the working out in Ms. Todd's case of the proposed theory for how the flu vaccine arguably could have injured her. Thus, there is no evidence that Petitioner possessed any of the primary autoantibodies that might arguably be associated with the kind of nerve function interference proposed by Petitioner—and she was tested for some. Ex. 6 at 20, 22, 24–25; Ex. 12 at 8.[24] There was no other indirect evidence of inflammation that would normally be associated with an autoimmune condition—and even the weak evidence of it was from too much later in her medical history post-vaccination to link it persuasively to the September 2012 vaccination. And, as already noted, the symptoms complained of by Petitioner, while suggestive of some kind of neuropathy, resisted a formal diagnosis, nor did her treaters ultimately conclude that her injury was autoimmune generally or vaccine-caused specifically. Whatever Petitioner suffers from, its nature and cause remains unidentified—and preponderantly has not been associated with her receipt of the flu vaccine.[25]

CONCLUSION

The Vaccine Act permits me to award compensation only if a Petitioner alleging a "non-Table Injury" can show by medical records or competent medical opinion that the injury was more likely than not vaccine-caused. Here, Petitioner has not established with preponderant evidence her proposed diagnosis, nor has she demonstrated the flu vaccine could bring it (or something comparable) about. I therefore **DENY** entitlement in this case.

In the absence of a timely-filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accord with this decision.[26]

[24] Although the evidence of successful immunosuppressive treatments might also be circumstantial evidence supporting Petitioner's autoimmune contentions, this must be weighed against both the affirmative absence of evidence of persistent inflammation in Petitioner, as well as the fact that treaters largely did not conclude her symptoms had an autoimmune origin.

[25] With respect to the third *Althen* prong, the onset of Petitioner's putatively neuropathic symptoms—two to three weeks post-vaccination—is not inconsistent with the timeframe for clinical manifestations of an autoimmune response after causal trigger and is also consistent with Petitioner's theory. However, the theory itself, as noted above, is insufficiently supported with reliable scientific or medical evidence—nor did Petitioner establish in the first place she had NLDSFN, or any other neuropathic injury for that matter. Regardless, my determination herein turns more on the first and second prongs, so Petitioner's success or failure at establishing evidence to support this one does not alter my conclusion.

[26] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by filing a joint notice renouncing their right to seek review.

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master